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(FILE 'HOME' ENTERED AT 14:17:57 ON 11 JUL 2005)

FILE 'MEDLINE' ENTERED AT 14:18:42 ON 11 JUL 2005

L1	759	S	EQUINE HERPESVIRUS
L2	68066	S	ATTENUATED
L3	33	S	L1 AND L2
L4	110497	S	PROMOTER
L5	3	S	L3 AND L4
L6	93	S	MUTATED PROMOTER
L7	0	S	L6 AND L1
L8	0	S	IE REGION AND L1
L9	2806	S	IMMEDIATE EARLY GENE
L10	22	S	L9 AND L1
L11	0	S	DHORE C R.AU.
		E	DHORE C R.AU.
		E	DHORE .AU.
		E	VISSER N .AU.
		E	VISSER N/AU
L12	28	S	E3
L13	0	S	L1 AND L12
L14	23	S	VIRUS AND L12
L15	130	S	ENDOGENOUS PROMOTER
L16	0	S	L1 AND L15
L17	58	S	L4 AND L1
L18	9	S	MUTANT AND L17

=> d l10 2 18 all

L10 ANSWER 2 OF 22 MEDLINE on STN
AN 2002208660 MEDLINE
DN PubMed ID: 11942126
TI EHV-1 gene63 is not essential for in vivo replication in horses and mice, nor does it affect reactivation in the horse: short communication.
AU Iqbal J; Purewal A S; Edington N
CS Department of Veterinary Basic Sciences and Department of Pathology and Infectious Diseases, Royal Veterinary College, Royal College Street, London NW1 0TU, UK.. jiqbal@rvc.ac.uk
SO Acta veterinaria Hungarica, (2001) 49 (4) 473-8.
Journal code: 8406376. ISSN: 0236-6290.
CY Hungary
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200204
ED Entered STN: 20020412
Last Updated on STN: 20020429
Entered Medline: 20020426
AB The aim of this study was to investigate the role of **immediate early gene** (gene63) in the pathogenesis of **equine herpesvirus** 1 (EHV-1) acute and latent infections in equine and murine models. EHV-1 gene63 mutant virus (g63mut) along with EHV-1 (Ab4) was used for intracerebral and intranasal infection of 3 and 17-day-old mice. Both viruses were recovered at the same frequency from tissues after infection. Two Welsh ponies were infected via the intranasal route with each of the viruses. Acute infection was monitored by virus isolation from nasal swabs and peripheral blood leukocytes. Six weeks post infection, peripheral blood leukocytes were taken from ponies and in vitro reactivation was positive for both viruses. At autopsy, both viruses were isolated by co-cultivation from bronchial and submandibular lymph nodes. These findings indicate that the mutation of EHV-1 gene63 does not play a role in the establishment and reactivation from latency.
CT Acute Disease
Animals
Disease Models, Animal
*Herpesviridae Infections: VE, veterinary
Herpesviridae Infections: VI, virology
Herpesvirus 1, Equid: GE, genetics
*Herpesvirus 1, Equid: GD, growth & development
Horses
Mice
Mice, Inbred BALB C
Research Support, Non-U.S. Gov't
*Viral Proteins: GE, genetics
Virus Latency
CN 0 (Viral Proteins); 0 (gene 63 protein, **Equine herpesvirus** 1)

L10 ANSWER 18 OF 22 MEDLINE on STN
AN 92114198 MEDLINE
DN PubMed ID: 1309921
TI Characterization of the regulatory functions of the **equine herpesvirus** 1 **immediate-early gene** product.
AU Smith R H; Caughman G B; O'Callaghan D J
CS Department of Microbiology and Immunology, Louisiana State University Medical Center, Shreveport 71130-3932.
NC AI 22001 (NIAID)
SO Journal of virology, (1992 Feb) 66 (2) 936-45.
Journal code: 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals

EM 199202
ED Entered STN: 19920308
Last Updated on STN: 19980206
Entered Medline: 19920214

AB Use of the translation-inhibiting drug cycloheximide has indicated that the **equine herpesvirus** 1 (EHV-1) immediate-early (IE) gene, the sole EHV-1 IE gene, encodes a major viral regulatory protein since IE mRNA translation is a prerequisite for all further viral gene expression (W.L. Gray, R. P. Baumann, A. T. Robertson, G. B. Caughman, D. J. O'Callaghan, and J. Staczek, Virology 158:79-87, 1987). An EHV-1 IE gene expression vector (pSVIE) in combination with chimeric EHV-1 promoter-chloramphenicol acetyltransferase (CAT) reporter constructs was used in transient transfection assays to characterize the regulatory functions of the IE gene product. These experiments demonstrated that (i) the EHV-1 IE gene product is a bifunctional protein capable of both positive and negative modulation of gene expression; (ii) the IE gene product possesses an autoregulatory function which represses the IE promoter; (iii) IE autoregulation is dependent on IE promoter sequences mapping within positions -288 to +73 relative to the transcription initiation site (+1) of the IE gene; (iv) the IE gene product can independently activate the EHV-1 tk promoter (an early promoter) by as much as 60-fold; (v) two EHV-1 beta-gamma (leaky late) promoters, those of IR5 (gene 5 in the inverted repeat) and the glycoprotein D gene, demonstrate a requirement for both the IE gene product as well as a gene product encoded within the EHV-1 XbaI G fragment for significant activation; and (vi) the IE gene product is capable of activating heterologous viral promoters.

CT Animals
Chloramphenicol O-Acetyltransferase: GE, genetics
Chloramphenicol O-Acetyltransferase: ME, metabolism
Cloning, Molecular
Gene Expression Regulation, Viral
*Genes, Viral
Genetic Vectors
*Herpesvirus 1, Equid: GE, genetics

114 9

L14 ANSWER 9 OF 23 MEDLINE on STN
AN 96040134 MEDLINE
DN PubMed ID: 7559856
TI A mouse model for testing the pathogenicity of equine herpes **virus** -1 strains.
AU van Woensel P A; Goovaerts D; Markx D; **Visser N**
CS Department of Virological Research and Development, Intervet International B.V., Boxmeer, Netherlands.
SO Journal of virological methods, (1995 Jul) 54 (1) 39-49.
Journal code: 8005839. ISSN: 0166-0934.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199511
ED Entered STN: 19951227
Last Updated on STN: 19951227
Entered Medline: 19951109

=> d l14 9 ab

L14 ANSWER 9 OF 23 MEDLINE on STN
AB A mouse model was developed for testing the pathogenicity of equine herpes **virus**-1 (EHV-1) strains. The model was validated with EHV-1 strains that are known to be of a low or high pathogenicity in horses. From all parameters tested, the safety index, which was calculated from the body weights of the mice after infection, proved to be the best predictive parameter. When this parameter was used, good and reliable correlations were found with the pathogenicity of the EHV-1 strains in horses. This method enabled the differentiation between the two experimental EHV-1 strains whose genetic backgrounds were supposedly equal.

=> d l18 1 2 9 all

L18 ANSWER 1 OF 9 MEDLINE on STN
AN 2004510715 MEDLINE
DN PubMed ID: 15479811
TI A negative regulatory element (base pairs -204 to -177) of the EICP0 **promoter** of **equine herpesvirus** 1 abolishes the EICP0 protein's trans-activation of its own **promoter**.
AU Kim Seong K; Albrecht Randy A; O'Callaghan Dennis J
CS Department of Microbiology and Immunology, Louisiana State University Health Sciences Center, 1501 Kings Highway, P.O. Box 33932, Shreveport, LA 71130-3932, USA.
NC AI-22001 (NIAID)
P20-RR018724 (NCRR)
SO Journal of virology, (2004 Nov) 78 (21) 11696-706.
Journal code: 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200411
ED Entered STN: 20041014
Last Updated on STN: 20041117
Entered Medline: 20041116
AB The early EICP0 protein is a powerful trans-activator that activates all classes of **equine herpesvirus** 1 (EHV-1) promoters but, unexpectedly, trans-activates its own **promoter** very weakly. Transient transfection assays that employed constructs harboring deletions within the EICP0 **promoter** indicated that EICP0 cis-acting sequences within bp -224 to -158 relative to the first ATG abolished the EICP0 protein's trans-activation of its own **promoter**. When inserted into the promoters of other EHV-1 genes, this sequence also downregulated activation of the immediate-early IE(-169/+73), early thymidine kinase TK(-215/+97), and late glycoprotein K gK(-83/+14) promoters, indicating that the cis-acting sequence (-224 to -158) downregulated expression of representative promoters of all classes of EHV-1 genes and contains a negative regulatory element (NRE). To define the cis-acting element(s), three synthetic oligonucleotides (Na [bp -224 to -195], Nb [bp -204 to -177], and Nc [bp -185 to -156]) were synthesized and cloned upstream of the EICP0(-157/-21) **promoter**. Of the three synthetic sequences, only the Nb oligonucleotide caused the downregulation of the EICP0 **promoter**. The NRE was identified as a 28-bp element to lie at -204 to -177 that encompassed the sequence of ([-204]AGATACAGATGTTCGATAAATTGGAACC[-177]). Gel shift assays performed with mouse L-M, rabbit RK-13, and human HeLa cell nuclear extracts and gamma-(32)P-labeled wild-type and **mutant** NREs demonstrated that a ubiquitous nuclear protein(s) (NRE-binding protein, NREBP) binds specifically to a sequence (bp -193 to -183) in the NRE. The NREBP is also present in the nucleus of EHV-1-infected cells; however, the amount of NREBP in EHV-1-infected L-M cells that bound to the Nb oligonucleotide was reduced compared to that in uninfected L-M cells. Transient transfection assays showed that deletions or mutations within the NREBP-binding site abolished the NRE activity of the EICP0 **promoter**. These results suggested that the NREBP may mediate the NRE activity of the EICP0 **promoter** and may function in the coordinate expression of EHV-1 genes.
CT Animals
Base Sequence
Hela Cells
*Herpesvirus 1, Equid: GE, genetics
Humans
Mice
Molecular Sequence Data
Nuclear Proteins: ME, metabolism
***Promoter Regions (Genetics)**
Rabbits
Research Support, U.S. Gov't, P.H.S.

*Trans-Activation (Genetics)
*Trans-Activators: GE, genetics
*Viral Regulatory Proteins: GE, genetics
CN 0 (Nuclear Proteins); 0 (Trans-Activators); 0 (Viral Regulatory Proteins)

L18 ANSWER 2 OF 9 MEDLINE on STN
AN 2003042997 MEDLINE
DN PubMed ID: 12552007
TI Interaction of the **equine herpesvirus** 1 EICP0 protein with the immediate-early (IE) protein, TFIIB, and TBP may mediate the antagonism between the IE and EICP0 proteins.
AU Kim Seong K; Jang Hyung K; Albrecht Randy A; Derbigny Wilbert A; Zhang Yunfei; O'Callaghan Dennis J
CS Department of Microbiology and Immunology, Louisiana State University Health Sciences Center, 1501 Kings Highway, Shreveport, LA 71130-3932, USA.
NC AI 22001 (NIAID)
SO Journal of virology, (2003 Feb) 77 (4) 2675-85.
Journal code: 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200303
ED Entered STN: 20030129
Last Updated on STN: 20030316
Entered Medline: 20030314
AB The **equine herpesvirus** 1 (EHV-1) immediate-early (IE) and EICP0 proteins are potent trans-activators of EHV-1 promoters; however, in transient-transfection assays, the IE protein inhibits the trans-activation function of the EICP0 protein. Assays with IE **mutant** proteins revealed that its DNA-binding domain, TFIIB-binding domain, and nuclear localization signal may be important for the antagonism between the IE and EICP0 proteins. In vitro interaction assays with the purified IE and EICP0 proteins indicated that these proteins interact directly. At late times postinfection, the IE and EICP0 proteins colocalized in the nuclei of infected equine cells. Transient-transfection assays showed that the EICP0 protein trans-activated EHV-1 promoters harboring only a minimal **promoter** region (TATA box and cap site), suggesting that the EICP0 protein trans-activates EHV-1 promoters by interactions with general transcription factor(s). In vitro interaction assays revealed that the EICP0 protein interacted directly with the basal transcription factors TFIIB and TBP and that the EICP0 protein (amino acids [aa] 143 to 278) mediated the interaction with aa 125 to 174 of TFIIB. Our unpublished data showed that the IE protein interacts with the same domain (aa 125 to 174) of TFIIB and with TBP. Taken together, these results suggested that interaction of the EICP0 protein with the IE protein, TFIIB, and TBP may mediate the antagonism between the IE and EICP0 proteins.
CT Animals
Fibroblasts
*Gene Expression Regulation, Viral
Herpesvirus 1, Equid: GE, genetics
*Herpesvirus 1, Equid: ME, metabolism
Horses
Humans
Immediate-Early Proteins: GE, genetics
*Immediate-Early Proteins: ME, metabolism
Mice
Promoter Regions (Genetics)
Research Support, U.S. Gov't, P.H.S.
TATA-Box Binding Protein: GE, genetics
TATA-Box Binding Protein: ME, metabolism
Trans-Activation (Genetics)
Trans-Activators: GE, genetics
*Trans-Activators: ME, metabolism
Transcription Factor TFIIB: GE, genetics
Transcription Factor TFIIB: ME, metabolism

Transfection
Tumor Cells, Cultured
Viral Proteins: GE, genetics
*Viral Proteins: ME, metabolism

CN 0 (Immediate-Early Proteins); 0 (TATA-Box Binding Protein); 0 (Trans-Activators); 0 (Transcription Factor TFIIB); 0 (Viral Proteins); 0 (gene 63 protein, **Equine herpesvirus** 1)

L18 ANSWER 9 OF 9 MEDLINE on STN
AN 97151083 MEDLINE
DN PubMed ID: 8995619
TI The ICP22 protein of **equine herpesvirus** 1 cooperates with the IE protein to regulate viral gene expression.
AU Kim S K; Holden V R; O'Callaghan D J
CS Department of Microbiology and Immunology, Louisiana State University Medical Center, Shreveport 71130-3932, USA.
NC AI-22001 (NIAID)
SO Journal of virology, (1997 Feb) 71 (2) 1004-12.
Journal code: 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199702
ED Entered STN: 19970305
Last Updated on STN: 19970305
Entered Medline: 19970218
AB The **equine herpesvirus** 1 (EHV-1) immediate-early (IE) phosphoprotein is essential for the activation of transcription from viral early and late promoters and regulates transcription from its own **promoter**. The EHV-1 EICP22 protein, a homolog of ICP22 of herpes simplex virus, increased the in vitro DNA binding activity of the IE protein for sequences in the IE, early, and late promoters. The EICP22 protein affected the rate as well as the extent of the IE protein binding to **promoter** DNA sequences. To study the DNA binding activity of the IE protein, Trp493, Gln495, Asn496, and Lys498 of the WLQN region, which is directly involved in DNA binding, were replaced with Ser (IEW493S), Glu (IEQ495E), Ile (IEN496I), and Glu (IEK498E), respectively. Gel shift assays revealed that the glutathione S-transferase (GST)-IEQ495E(407-615) and GST-IEK498E(407-615) proteins failed to bind to the IE **promoter**, indicating that the Gln and Lys residues are important for the DNA binding activity. In the presence of the GST-EICP22 protein, DNA binding activity of the GST-IEQ495E(407-615) protein was restored, suggesting that the EICP22 protein cooperates with the IE protein to regulate EHV-1 gene expression. Transient-transfection assays also showed that the EICP22 protein allowed the IEQ495E **mutant** to be functional as a transactivator. These results are unique and may represent an important role for the EICP22 protein in EHV-1 gene regulation.
CT *Gene Expression Regulation, Viral
*Herpesvirus 1, Equid: GE, geneti

*Viral Proteins: GE, genetics
CN 0 (DNA, Viral); 0 (Viral Proteins); 0 (gene 63 protein, **Equine herpesvirus** 1)

L5 ANSWER 3 OF 3 MEDLINE on STN
AN 97288320 MEDLINE
DN PubMed ID: 9143298
TI An **equine herpesvirus**-1 gene 71 deletant is **attenuated** and elicits a protective immune response in mice.
AU Marshall K R; Sun Y; Brown S M; Field H J
CS MRC Virology Unit, Glasgow, United Kingdom.
SO Virology, (1997 Apr 28) 231 (1) 20-7.
Journal code: 0110674. ISSN: 0042-6822.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199706
ED Entered STN: 19970612
Last Updated on STN: 19970612
Entered Medline: 19970604
AB The pathogenesis of pulmonary infection and the immune response following intranasal inoculation of mice with two **equine herpesvirus** type 1 (EHV-1) deletion mutants have been assessed. The mutants, ED71 and ED75, have deletions in genes 71 (EUS4) and 75 (10K), respectively. Deletions were replaced by the Escherichia coli lacZ gene driven by the simian virus 40 (SV40) early **promoter**. It has previously been shown that the protein products of genes 71 and 75 are dispensable in vitro but that removal of gene 71 results in a defect in virus maturation and capsid envelopment which impairs the ability of mutant virus to spread via release and readsorption. This study demonstrated that the 192-kDa gene 71 product is required for full expression of virulence in mice, whereas the putative 10-kDa product of gene 75 has minimal effect. Both mutants exhibited the same tissue and cytotropism as wild-type EHV-1 and induced both humoral and cell-mediated immune responses indistinguishable from those induced by the parental strain. Irrespective of the reduced pathogenicity of the gene 71 mutant, infected mice were protected against a challenge with wild-type EHV-1. These findings highlight the potential of ED71 as a vaccine candidate.
CT Check Tags: Female
Animals
Gene Deletion
Genes, Viral

d 15 2-3 all

L5 ANSWER 2 OF 3 MEDLINE on STN
AN 97332318 MEDLINE
DN PubMed ID: 9188552
TI The ICP0 protein of **equine herpesvirus** 1 is an early protein that independently transactivates expression of all classes of viral promoters.
AU Bowles D E; Holden V R; Zhao Y; O'Callaghan D J
CS Department of Microbiology and Immunology, Louisiana State University Medical Center, Shreveport 71130-3932, USA.
NC AI-22001 (NIAID)
SO Journal of virology, (1997 Jul) 71 (7) 4904-14.
Journal code: 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-U81154
EM 199707
ED Entered STN: 19970721
Last Updated on STN: 19970721
Entered Medline: 19970710
AB To assess the role of the **equine herpesvirus** type 1 (EHV-1) ICP0 protein (EICP0) in gene regulation, a variety of molecular studies on the EICP0 gene and gene products of both the **attenuated** cell culture-adapted Kentucky A (KyA) strain and the Ab4p strain were conducted. These investigations revealed that (i) the ICP0 open reading frame (ORF) of the KyA virus strain is 1,257 bp in size and would encode a protein of 419 amino acids, and in comparison to the ICP0 gene (ORF63) of the Ab4p strain of 1,596 bp (E. A. Telford, M. S. Watson, K. McBride, and A. J. Davison, Virology 189:304-316, 1992), it has an internal in-frame deletion of 339 bp; (ii) one early transcript of 1.4 kb predicted to encode the EICP0 protein and a late transcript of 1.8 kb are detected in Northern blot analyses using probes containing the EICP0 ORF; (iii) the KyA EICP0 protein (50 kDa) and the Ab4p EICP0 protein (80 kDa) are expressed as several species of early proteins that are first detected at 3 to 4 h postinfection by Western blot analyses of infected-cell polypeptides, using an antiserum generated to a TrpE fusion protein that harbors amino acids 46 to 153 of the EICP0 protein; and (iv) the EICP0 protein of both EHV-1 strains is a potent transactivator of EHV-1 genes. Transient expression assays using a simian virus 40 expression construct of the EICP0 protein of the KyA strain showed that the EICP0 protein independently transactivated chloramphenicol acetyltransferase reporter constructs under the control of the immediate-early **promoter** (3.9-fold), the early thymidine kinase **promoter** (95-fold), the late (gamma1) IR5 **promoter** (85-fold), and the late (gamma2) glycoprotein K **promoter** (21-fold). The finding that the EICP0 protein of the KyA virus can function as an activator of gene expression indicates that amino acids corresponding to residues 319 to 431 of the Ab4p EICP0 protein are not essential for EICP0 transactivation of EHV-1 promoters.
CT Amino Acid Sequence
Animals
Base Sequence
Cell Line
DNA, Viral
*Gene Expression Regulation, Viral
Genes, Viral
Molecular Sequence Data
***Promoter Regions (Genetics)**
Rabbits
Research Support, U.S. Gov't, Non-P.H.S.
Research Support, U.S. Gov't, P.H.S.
Sequence Analysis, DNA
Sequence Homology, Amino Acid
Sequence Homology, Nucleic Acid
*Trans-Activation (Genetics)

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DATE: Monday, July 11, 2005

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		DB=PGPB; PLUR=YES; OP=ADJ	
☐	L20	DHORE.in.	0
		DB=USPT; PLUR=YES; OP=ADJ	
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		DB=DWPI; PLUR=YES; OP=ADJ	
☐	L18	DHORE C R.in.	1
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☐	L16	EP-668355-A1.did.	1
☐	L15	WO-9804286-A2.did.	1
☐	L14	WO-9804286-A2.did.	1
☐	L13	WO-9804286-A2.did.	1
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☐	L11	WO-200209750-A2.did.	0
		DB=DWPI; PLUR=YES; OP=ADJ	
☐	L10	equine herpesvirus	16
		DB=PGPB; PLUR=YES; OP=ADJ	
☐	L9	equine herpesvirus.clm.	10
☐	L8	equine herpesvirus	144
		DB=DWPI; PLUR=YES; OP=ADJ	
☐	L7	mutated equine herpesvirus	0
		DB=PGPB; PLUR=YES; OP=ADJ	
☐	L6	mutated equine herpesvirus	0
		DB=USPT; PLUR=YES; OP=ADJ	
☐	L5	equine herpesvirus and Salimi.xp.	15
☐	L4	mutated equine herpesvirus	0
☐	L3	attenuated equine herpesvirus	3
☐	L2	L1 and IE gene	6
	L1	equine herpesvirus	257

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw D

☐ 3. Document ID: US 5674499 A

L3: Entry 3 of 3 File: USPT Oct 7, 1997

US-PAT-NO: 5674499
DOCUMENT-IDENTIFIER: US 5674499 A
** See image for Certificate of Correction **

TITLE: Equine herpesvirus gene 15 mutants

DATE-ISSUED: October 7, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Willemse; Martha Jacoba	Nijmegen			NL
Sondermeijer; Paulus Jacobus Antonius	Boxmeer			NL
Nicolson; Lesley	Glasgow			GB6

US-CL-CURRENT: 424/199.1; 424/205.1, 424/229.1, 435/235.1, 435/252.3, 435/320.1, 435/325, 536/23.72

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw D

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Terms	Documents
attenuated equine herpesvirus	3

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☐ 1. Document ID: US 6803041 B2

L3: Entry 1 of 3 File: USPT Oct 12, 2004

US-PAT-NO: 6803041
DOCUMENT-IDENTIFIER: US 6803041 B2
** See image for Certificate of Correction **

TITLE: Equine herpesvirus vaccine

DATE-ISSUED: October 12, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Mellencamp; Mark W.	St. Joseph	MO		

US-CL-CURRENT: 424/229.1; 424/202.1, 424/204.1, 424/205.1, 424/206.1, 424/278.1, 424/280.1, 435/173.3, 435/235.1, 435/236, 435/237, 435/238

Full | Title | Citation | Front | Review | Classification | Date | Reference | | | Claims | KWIC | Draw D

☐ 2. Document ID: US 6187320 B1

L3: Entry 2 of 3 File: USPT Feb 13, 2001

US-PAT-NO: 6187320
DOCUMENT-IDENTIFIER: US 6187320 B1

TITLE: Equine herpesviruses (EHV) which contain foreign DNA, process for the preparation thereof and the use thereof in vaccines

DATE-ISSUED: February 13, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Darai; Gholamreza	Heidelberg			DE
Thein; Peter	Oberzeitlbach			DE
Strube; Walter	Koln			DE
Ludwig; Hanns	Berlin			DE

US-CL-CURRENT: 424/229.1; 435/235.1, 435/236, 435/320.1, 530/350, 536/23.72

(1) an immunogenic composition comprising (I);

(2) a vaccine composition comprising (I); and

(3) determining (II) the non-pathogenicity of an EHV-1 virus present in a horse subject previously administered with a non-pathogenic EHV-1 isolate comprising a mutation in the IE gene, by isolating the virus from the subject:

(a) detecting the presence of the mutant IE protein of the non-pathogenic isolate and the absence of a wild type IE protein in the virus; or

(b) detecting the absence in the serum of the subject of an antibody specific for the deleted portion for the IE protein; or

(c) detecting the absence of the wild-type IE nucleotide sequence and the presence of the mutant IE sequence; or

(d) determining the temperature sensitivity of the virus as identical to that of the non-pathogenic EHV-1 isolate, to determine the virus as non-pathogenic.

ACTIVITY - Virucide; immunostimulant.

Mutant viruses KyAd644/824, KyAn1411, KyAin1411 and KyAE34Q were tested in mice. Mice were anesthetized with halothane and inoculated intranasally with 2 x 106 plaque forming units (PFU) of EHV-1 Kya or a mutant virus in a volume of 50 mu l. Control mice received 50 mu l of culture medium alone. Immunized mice were monitored daily for development of clinical signs of EHV-1 infection such as ruffled fur, loss of body weight, labored breathing, lethargy and huddling. No clinical disease was observed with mice infected with any of the four mutant viruses tested. To assess primary cytotoxic T lymphocyte (CTL) responses, lymphocytes were isolated from the mediastinal lymph nodes (MLN) 5 days postinoculation, and a single-cell suspension was obtained. Cytolytic activity was assessed. As indicated, all four mutant viruses tested, KyAd644/824, KyAn1411, KyAin1411 or KyAE34Q, induced a CTL response at a level similar to that induced by parent KyA virus.

MECHANISM OF ACTION - Vaccine.

USE - (I) is useful for stimulating an immune response, preferably a cell-mediated or humoral immune response, against EHV-1, and for preventing or inhibiting an EHV-1 infection in a horse (claimed).

ABSTRACTED-PUB-NO: WO 200209750A
EQUIVALENT-ABSTRACTS:

CHOSEN-DRAWING: Dwg.0/5

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L10: Entry 7 of 16 File: DWPI Sep 1, 2003

DERWENT-ACC-NO: 2002-206153
DERWENT-WEEK: 200465

TITLE: Novel mutant equine herpesvirus type-1 isolates having mutation in immediate early gene, useful in formulating vaccine compositions for preventing and treating equine herpesvirus type-1 infections in horses

INVENTOR: O'CALLAGHAN, D J; OCALLAGHAN, D J

PRIORITY-DATA: 2000US-0626748 (July 27, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
MX 2003000823 A1	September 1, 2003		000	A61K039/12
WO 200209750 A2	February 7, 2002	E	068	A61K039/12
AU 200182997 A	February 13, 2002		000	A61K039/12
EP 1317481 A2	June 11, 2003	E	000	C07K014/03
JP 2004512826 W	April 30, 2004		109	C12N015/09
NZ 523772 A	June 25, 2004		000	A61K039/12

INT-CL (IPC): A61 K 39/12; A61 K 39/245; A61 K 39/27; A61 P 31/22; C07 K 14/03; C12 N 7/00; C12 N 15/09; C12 Q 1/68; C12 Q 1/70; G01 N 27/447; G01 N 33/483; G01 N 33/53; G01 N 33/569

ABSTRACTED-PUB-NO: WO 200209750A
BASIC-ABSTRACT:

NOVELTY - A mutant equine herpesvirus type-1 (EHV-1) isolate (I), in particular a replication-competent EHV-1 isolate comprising a mutation in the immediate-early (IE) gene of the viral genome, is new.

DETAILED DESCRIPTION - A mutant equine herpesvirus type-1 (EHV-1) isolate (I), in particular a replication-competent EHV-1 isolate comprising a mutation in the immediate-early (IE) gene of the viral genome, is new.

(I) comprises a mutation chosen from deletion mutations, Delta SRT1, Delta SRT2, d178/627, d552/897, d644/824; nonsense mutations n627, n951, n1029, n1411; insertion mutations in628, in1411; and point mutations D20N, D24N, L12P, L12E, F15D, E34Q.

INDEPENDENT CLAIMS are also included for the following: